08/832,443

********** STN Columbus **********

FILE 'HOME' ENTERED AT 14:26:59 ON 16 NOV 1999

=> file medline cancerlit biosis embase scisearch

FILE 'MEDLINE' ENTERED AT 14:28:40 ON 16 NOV 1999

FILE 'CANCERLIT' ENTERED AT 14:28:40 ON 16 NOV 1999

FILE 'BIOSIS' ENTERED AT 14:28:40 ON 16 NOV 1999
COPYRIGHT (C) 1999 BIOSIS(R)

FILE 'EMBASE' ENTERED AT 14:28:40 ON 16 NOV 1999
COPYRIGHT (C) 1999 Elsevier Science B.V. All rights reserved.

FILE 'SCISEARCH' ENTERED AT 14:28:40 ON 16 NOV 1999
COPYRIGHT (C) 1999 Institute for Scientific Information (ISI) (R)

=> s inprol

L1 2 INPROL

=> d l1 1-2 ibib ab

L1 ANSWER 1 OF 2 EMBASE COPYRIGHT 1999 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 86057720 EMBASE
DOCUMENT NUMBER: 1986057720
TITLE: [Disko-radicular conflict treatment by intradiscal chymopapain].
TRAITEMENT DES CONFLITS DISCO-RADICULAIRES PAR INJECTION INTRADISCALE D'APROTININE.
AUTHOR: Amor B.; Revel M.; Dougados M.; et al.
CORPORATE SOURCE: Clinique de Rhumatologie, Hopital Cochin, 75014 Paris, France
SOURCE: Medecine et Armees, (1985) 13/8 (751-754).
CODEN: MDARC4
COUNTRY: France
DOCUMENT TYPE: Journal
FILE SEGMENT: 037 Drug Literature Index
LANGUAGE: French

L1 ANSWER 2 OF 2 EMBASE COPYRIGHT 1999 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 78330046 EMBASE
DOCUMENT NUMBER: 1978330046
TITLE: [Pulmonary embolism].
EMBOLIES PULMONAIRES. SIGNES, DIAGNOSTIC, TRAITEMENT.
AUTHOR: Roudaut R.
CORPORATE SOURCE: France
SOURCE: Bordeaux Medical, (1978) 11/12 (1061-1066).
CODEN: BOMEBE
COUNTRY: France
DOCUMENT TYPE: Journal
FILE SEGMENT: 037 Drug Literature Index
015 Chest Diseases, Thoracic Surgery and Tuberculosis
006 Internal Medicine
LANGUAGE: French

=> s fphfdlshgsaqvs or phe-pro-his-phe-asp-leu-ser-his-gly-ser-ala-gln-val

L2 0 FPHFDLSHGSAQVS OR PHE-PRO-HIS-PHE-ASP-LEU-SER-HIS-GLY-SER-ALA-GLN-VAL

=> hemoglobin and (stem cell or hematopoie? or progenitor(w)cell or pluripotent?)

HEMOGLOBIN IS NOT A RECOGNIZED COMMAND
The previous command name entered was not recognized by the system.
For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).

=> s hemoglobin and (stem cell or hematopoie? or progenitor(w)cell or pluripotent?)

L3 3111 HEMOGLOBIN AND (STEM CELL OR HEMATOPOIE? OR PROGENITOR(W)CELL OR PLURIPOTENT?)

=> s l3 and (stimulat? or prolifer?)

L4 1002 L3 AND (STIMULAT? OR PROLIFER?)

=> dup rem

ENTER L# LIST OR (END):l4

PROCESSING IS APPROXIMATELY 40% COMPLETE FOR L4
PROCESSING COMPLETED FOR L4
L5 466 DUP REM L4 (536 DUPLICATES REMOVED)

=> s l3 and (inhib? or reduc? or abrogat? or antagon?)

3 FILES SEARCHED...
L6 790 L3 AND (INHIB? OR REDUC? OR ABROGAT? OR ANTAGON?)

=> dup rem

ENTER L# LIST OR (END):l6

PROCESSING IS APPROXIMATELY 87% COMPLETE FOR L6
PROCESSING COMPLETED FOR L6
L7 381 DUP REM L6 (409 DUPLICATES REMOVED)

=> s hemoglobin and ((stem cell or hematopoie? or progenitor(w)cell or pluripotent?)5a(inhib? or reduc? or abrogat? or antagon?))

MISSING OPERATOR RIPOTENT?)5A
The search profile that was entered contains terms or nested terms that are not separated by a logical operator.

=> s hemoglobin and ((stem cell or hematopoie? or progenitor(w)cell or pluripotent?) 5a (inhib? or reduc? or abrogat? or antagon?))

MISSING OPERATOR IPOTENT?) 5A
The search profile that was entered contains terms or nested terms that are not separated by a logical operator.

=> s hemoglobin 5a ((stem cell or hematopoie? or progenitor(w)cell or pluripotent?) and (inhib? or reduc? or abrogat? or antagon?))

MISSING OPERATOR '5A ((STEM'
The search profile that was entered contains terms or nested terms that are not sep

=> s hemoglobin and ((stem ce
pluripotent?) (5a) (inhib? or re

2 FILES SEARCHED...
4 FILES SEARCHED...
L8 80 HEMOGLOBIN AN
PROGENITOR(W)CELL
OR PLURIPOTENT?)
ANTAGON?)
)

=> dup rem

ENTER L# LIST OR (END):l8

PROCESSING COMPLETED F
L9 31 DUP REM L8 (49

=> d l9 1-31 ibib ab

L9 ANSWER 1 OF 31 BIOSI
ACCESSION NUMBER: 1999
DOCUMENT NUMBER: PRE
TITLE: **Hemoglobin** al
for **inhibiting stem** proliferation.
AUTHOR(S): Tsyrlova, I
CORPORATE SOURCE: Biote
Standards and Technology, Gaithe
ASSIGNEE: Pro-N
PATENT INFORMATION: US
SOURCE: Official Gaze
Trademark Office Patents, (A
PAGINATION.
ISSN: 0098-1133
DOCUMENT TYPE: Patent
LANGUAGE: English

L9 ANSWER 2 OF 31 MEDL
ACCESSION NUMBER: 1998
DOCUMENT NUMBER: 9844
TITLE: [A case of pos
dyserythropoiesis
lupus erythematos
Une observation d
dyserythropoi`ese
du lupus erythema
AUTHOR: Kettaneh A
J
L; Roux S; Kahn M
CORPORATE SOURCE: Ser
Bernard, Paris, France.
SOURCE: REVUE DE M
Journal code: SG
PUB. COUNTRY: France
Journal; Article;
LANGUAGE: French
FILE SEGMENT: Priority

08/832,443

ENTRY MONTH: 199901
ENTRY WEEK: 19990104
AB INTRODUCTION: Amongst the various causes of anemia in systemic lupus
erythematosus, isolated and acquired erythrocyte dysplasia is rare and most often part of global dysmyelopoiesis. EXEGESIS: The authors report a
case of acquired erythrocyte dysplastic syndrome that occurred in a 34-year-old woman in whom previous diagnosis had evidenced systemic lupus
erythematosus of rather benign course. Other causes of dysmyelopoiesis were ruled out. Myeloid **stem cell** cultures showed
selective **inhibition** of erythroid cells growing, with no particular effect of the patient's serum. While a corticosteroid treatment
with prednisone (1 mg/kg/d) did not show any efficacy upon anemia, the patient's pregnancy was followed by prolonged correction of
hemoglobin, making possible the tapering of prednisone down to 10 mg/d. CONCLUSION: Acquired erythrocyte dysplastic syndrome remains a rare
cause of anemia in systemic lupus erythematosus. This case report suggests
an immunological phenomenon, but the mechanisms underlying both the appearance and long-lasting remission after pregnancy remain unexplained.

L9 ANSWER 3 OF 31 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 97368455 MEDLINE
DOCUMENT NUMBER: 97368455
TITLE: Morphological changes and apoptosis in bone marrow from patients with myelodysplastic syndromes treated with granulocyte-CSF and erythropoietin [see comments].
COMMENT: Comment in: Leuk Res 1997 May;21(5):427-8
AUTHOR: Hellstrom-Lindberg E; Kanter-Lewensohn L; Ost A
CORPORATE SOURCE: Department of Hematology, Huddinge University Hospital,
Stockholm, Sweden.
SOURCE: LEUKEMIA RESEARCH, (1997 May) 21 (5) 415-25.
Journal code: K9M. ISSN: 0145-2126.
PUB. COUNTRY: ENGLAND: United Kingdom
(CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(MULTICENTER STUDY)
(RANDOMIZED CONTROLLED TRIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals; Cancer Journals
ENTRY MONTH: 199710
ENTRY WEEK: 19971002
AB A study of bone marrow morphology and apoptosis was undertaken in 51 patients with myelodysplastic syndromes (MDS) treated with granulocyte-colony-stimulating factor (G-CSF) and erythropoietin (EPO). In 19 of these
patients (37%), a significant improvement in the **hemoglobin** level was found after treatment. Apoptosis was measured using a nick-end labeling (TUNEL) technique. Patients with MDS had a significantly higher percentage of labelled (apoptotic) cells in the bone marrow compared to healthy individuals (56.3 +/- 3.8% vs. 16.2 +/- 1.4%, p = 0.0001). Patients with RAS showed a lower percentage of apoptotic cells than patients with RA (68.5 +/- 9% vs. 46.5 +/- 4.8%, p < 0.05), while patients with RAEB did not differ significantly from either RA or RAS. In the patients who responded to treatment, the bone marrow samples displayed significant morphological changes. The percentages of erythroid precursors
and myeloblasts were reduced after treatment, and patients who had ring sideroblasts before treatment also showed a reduction in the percentage of
these cells. Total erythroid index also decreased in responding patients. The percentage of apoptotic cells decreased significantly in responding patients (58.8 +/- 4.8% before treatment vs. 44.5 +/- 5.5% after treatment, mean reduction 18.3%, p = 0.0003), whereas no significant change was found in non-responding patients. Our results suggest that one important mechanism behind the positive effects of treatment with G-CSF
and EPO is a **reduction** in the degree of ineffective **hematopoiesis** in MDS.

L9 ANSWER 4 OF 31 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 97434708 MEDLINE
DOCUMENT NUMBER: 97434708
TITLE: Effect of successful parathyroidectomy on hematopoietic progenitor cells and parameters of red blood cells in patients with primary hyperparathyroidism.
AUTHOR: Kotzmann H; Abela C; Heindl J; Clodi M; Riedl M; Barnas U; Heinzl H; Niederle B; Geissler K; Waldhausl W; Luger A
CORPORATE SOURCE: Department of Medicine III, University of Vienna, Austria.
SOURCE: HORMONE AND METABOLIC RESEARCH, (1997 Aug) 29 (8) 387-92.
Journal code: GBD. ISSN: 0018-5043.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199801
ENTRY WEEK: 19980104
AB Elevated levels of parathyroid hormone (PTH) in primary and secondary hyperparathyroidism **inhibit hematopoiesis** at the level
of **hematopoietic** progenitor cells, mainly the burst forming units-erythroid (BFUe). Removal of parathyroid adenomas is associated with
an increase in hematopoietic progenitor cells. In contrast, a certain amount of PTH and calcium is needed to correct anemia after bleeding demonstrating that PTH has also a stimulatory effect on the bone marrow.
We examined the effect of parathyroidectomy (PTX) in 10 patients with histologically proven primary hyperparathyroidism on hematopoietic progenitor cells and several parameters of red blood cells before and at 5, 30 and 90 days after PTX. After successful surgery serum levels of iPTH
($p < 0.01$) and calcium ($p < 0.001$) decreased significantly. Subsequently a steady increase in all hematopoietic progenitor cell classes was observed reaching significance for BFUe only ($p < 0.05$). Red blood cells and **hemoglobin** reached nearly pretreatment values within 90 days after PTX after they had decreased due to surgery associated blood loss. 8 patients undergoing hemithyroidectomy without PTX showed a similar decrease in red blood cells and **hemoglobin** followed by a rise after the operation. The changes of these parameters did not differ significantly from the patients with pHPT. In contrast to the patients with pHPT, no changes in hematopoietic progenitor cells during the 90 days
were observed. The presented data provide further evidence that increased
PTH concentrations might **inhibit hematopoiesis** in humans in vivo. The **inhibition** can be reversed following PTX by normalisation of PTH concentrations.

L9 ANSWER 5 OF 31 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 97283967 MEDLINE
DOCUMENT NUMBER: 972[illegible]
TITLE: Hematological
with total body fa[illegible]
AUTHOR: Lambert M.[illegible]
Thissen J
P; Nagant de Deux[illegible]
CORPORATE SOURCE: Divisi[illegible] Medici[illegible]
University
Hospital, Universit[illegible]
Belgium.
SOURCE: INTERNATI[illegible]
(1997 May) 21
(4) 329-34
Journal code: BRF[illegible]
PUB. COUNTRY: United St[illegible]
Journal; Article; (
LANGUAGE: English
FILE SEGMENT: Priority
ENTRY MONTH: 199708
ENTRY WEEK: 1997080[illegible]
AB OBJECTIVE: To assess t[illegible] between tota[illegible]
(FM).
and hematological abnorma[illegible] nervosa (AN, N
Peripheral blood parameter[illegible] sition were [illegible]
anorectic patients and 19 a[illegible] ched healthy [illegible]
patients with AN, magnetic[illegible] (MRI) stu[illegible]
marrow
were also performed. RES[illegible] th controls[illegible]
AN had
-41% body weight, -81% FM[illegible] mass (LTM
bone
mineral content; they also [illegible] tal leukocyte[illegible]
vs. 6.28 +/- 0.33 x 10(3)/m[illegible] neutrophil[illegible]
vs. 3.46 +/- 0.20 x 10(3)/m[illegible] monocyte ([illegible]
vs. 0.37 +/- 0.03 x 10(3)/m[illegible] and platelet[illegible]
+/- 17 vs. 238 +/- 9 x 10(3)[illegible] Hemoglobin
level was normal and compar[illegible] In patien[illegible]
not in controls, total leuko[illegible] eosinophil, ar[illegible]
counts as well as **hemoglobin** [illegible] correlate[illegible]
FM expressed in absolute [illegible] ntage of body[illegible]
with LTM. Moreover, AN p[illegible] ntensity p[illegible]
suggestive
of serous atrophy of bone [illegible] not only l[illegible]
leukocyte, neutrophil, and [illegible] they also[illegible] N
than AN patients with norm[illegible] DISCUSSI[illegible]
Hematological
changes in AN, as assessed[illegible] parameter[illegible]
patterns
of bone marrow, are corre[illegible] FM depri[illegible]
that the **reduction** of adip[illegible] affects
hematopoiesis.

L9 ANSWER 6 OF 31 MEDL[illegible] DUPLICA
ACCESSION NUMBER: 964[illegible]
DOCUMENT NUMBER: 964[illegible]
TITLE: Adverse effe[illegible] protectior
prechemotherapy
granulocyte colon[illegible] support [illegible]
comments]
COMMENT: Comment [illegible] 1997 M[illegible]
AUTHOR: de Wit R; Ve[illegible] M Knuit [illegible]
Schmitz P I; Sto[illegible]
CORPORATE SOURCE: Dep[illegible]
Institute,

08/832,44 :

The Netherlands.
SOURCE: JOURNAL OF THE NATIONAL CANCER INSTITUTE, (1996 Oct 2) 88
(19) 1393-8
Journal code: J9J. ISSN: 0027-8874.
PUB. COUNTRY: United States
(CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LANGUAGE: English
FILE SEGMENT: Cancer Journals; Priority Journals
ENTRY MONTH: 199612
AB BACKGROUND: Increased proliferation of endogenous bone marrow progenitor
cells in response to the administration of **hematopoietic** growth factors, followed by **reduced** cell cycling or entrance of the cells into a quiescent state upon withdrawal of the growth factors, may have clinically relevant effects on the tolerance of the hematopoietic system to subsequent myelotoxic treatments. PURPOSE: We investigated the
ability of granulocyte colony-stimulating factor (G-CSF) to protect progenitor cells in the bone marrow of cancer patients from the toxic effects of subsequent treatments with chemotherapeutic agents.
METHODS:
Thirty-six patients with histologically documented, locally advanced or metastatic breast cancer were randomly assigned to receive doxorubicin once every 3 weeks at a dose of 75 mg/m2 and cyclophosphamide at a dose of
1000 mg/m2, with G-CSF administered either before and after chemotherapy
(18 patients) or after chemotherapy only (18 patients). For prechemotherapy administration of G-CSF, recombinant human methionyl (r-met Hu) G-CSF was administered subcutaneously to patients twice per day
for 5 days at a dose of 5 micrograms/kg, with the last dose given 48 hours
before the start of chemotherapy. For postchemotherapy administration of
G-CSF, r-met Hu G-CSF was administered subcutaneously to patients once per
day for 7 days at a dose of 5 micrograms/kg, with the first dose given 24 hours after chemotherapy. RESULTS: The incidence or the duration of
grade
4 neutropenia was not reduced in all patients by the use of prechemotherapy G-CSF; the incidence over all cycles of chemotherapy was
74% for patients treated with prechemotherapy and postchemotherapy G-CSF
and 66% for patients treated with postchemotherapy G-CSF only (two-sided
P, adjusted for dose = .21) and the median duration in both treatment arms
was 3 days (two-sided P = .19). Unexpectedly, the incidence of grades 3 and 4 thrombocytopenia was much greater in patients who received prechemotherapy G-CSF compared with those who did not (54% versus 6%,
respectively, over all chemotherapy cycles; two-sided P, adjusted for dose
< .001). No difference in the decrease in **hemoglobin** level
(adjusted for red blood cell transfusions) between patients in the two treatment arms was observed. CONCLUSIONS AND IMPLICATIONS: No beneficial
effects were associated with the administration of G-CSF to cancer patients prior to chemotherapy. The observation of more severe thrombocytopenia in patients treated with prechemotherapy G-CSF led us to
conclude that the proliferation of progenitor cells was still increased 48 hours after the last dose of G-CSF and that the administration of chemotherapy at or within this time period actually worsens the toxic effects on bone marrow. This result has important ramifications for the design of clinical cancer treatment protocols, especially those that involve shortened intervals between cycles of chemotherapeutic agent administration.

L9 ANSWER 7 OF 31 MEDLINE DUPLICATE 6
ACCESSION NUMBER: 96374369 MEDLINE
DOCUMENT NUMBER: 96374369
TITLE: A randomized, double-blind comparison of donor tolerance of 400 mL, 200 mL, and sham red cell donation.
AUTHOR: Smith K J; James D S; Hunt W C; McDonough W; Quintana R
CORPORATE SOURCE: Department of Pathology, University of New Mexico School of
Medicine, Albuquerque, USA.
SOURCE: TRANSFUSION, (1996 Aug) 36 (8) 674-80.
Journal code: WDN. ISSN: 0041-1132.
PUB. COUNTRY: United States
(CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199612
AB BACKGROUND: Volume replacement could allow the safe collection of twice
the normal amount of red cells in a standard donation. Studies in small numbers of donors have shown that a temporary decrease in red cell mass is
well tolerated when donors give twice the usual amount (170-225 mL) of red
cells in a standard 405- to 495-mL donation. Sham-donation control groups have not been included in previous studies of increased red cell donation, and perceptions of donation effects could have been biased. STUDY DESIGN
AND METHODS: In the study reported here, 17 male and 13 female volunteers
were randomly assigned to make a sham donation, 1-unit donation, or 2-unit
donation on an automated blood cell separator. Donor tolerance was assessed by ambulatory heart rate monitoring and by a poststudy interview.
Hemoglobin, hematocrit, ferritin, serum iron, total iron-binding capacity, red cell 2,3 DPG, and serum erythropoietin were measured before
and after donation for comparison of the erythropoietic responses in the three study groups. RESULTS: Red cells collected totaled 206 +/- 10 mL in
the 1-unit group and 414 +/- 21 mL in the 2-unit group. Changes in heart rate, systolic blood pressure, and diastolic blood pressure with donation and changes in heart rate recorded by ambulatory monitoring did not differ
for the experimental groups. Postdonation changes from baseline values were evaluated on Days 2, 7, and 14. Changes in **hemoglobin** were significantly different between groups ($p < 0.017$) in all postdonation tests. There were differences between groups in erythropoietin response,
red cell 2,3 DPG, ferritin levels, and **hemoglobin** synthesis.
Hemoglobin synthesis and mean changes in 2,3 DPG, erythropoietin, ferritin, and postdonation he
group than in the 1-unit gro
donations of 414 +/- 21 ml
450-mL blood donation, due
or
sham donations. Physiologic
response to **reduced** red ce
group, but the donation of
symptoms of reduced oxyge

L9 ANSWER 8 OF 31 MEDL
ACCESSION NUMBER: 964
DOCUMENT NUMBER: 964
TITLE: A semiautomat
stem cell suspensio
autotransplantati
AUTHOR: Ayello J; He
CORPORATE SOURCE: Depar
Surgeons, Columb
SOURCE: JOURNAL O
Journal code: B3
PUB. COUNTRY: United S
Journal; Article; (
LANGUAGE: English
FILE SEGMENT: Priority
ENTRY MONTH: 199612
AB Infusion of thawed cryo
associated
with a variety of complicat
(DMSO) and free **hemoglob**
DMSO is not removed befo
exposure of the cells to DM
describe a simple technique
DMSO content of bone ma
(PBSC)
suspensions. Sixty-five pa
volume-reduced SC cryopr
Semiautomated
volume reduction was perf
median
volumes of cryopreserved
PBSC
products and the mixed po
volume of SC infused was
differences
in cell recoveries between
demonstrated
minimal side effects durin
cancer patients underwent
priming and subsequent au
neutrophil count > 500/mi
median time to a platelet
6-18 days). Volume reduct
failure was performed sim
infusion.

L9 ANSWER 9 OF 31 MEDL
ACCESSION NUMBER: 954
DOCUMENT NUMBER: 954
TITLE: Myelodysplas
AUTHOR: Locatelli F
Severi F
CORPORATE SOURCE: Clin
Policlinico

08/832,443

San Matteo, Italy..
SOURCE: HAEMATOLOGICA, (1995 May-Jun) 80 (3) 268-79. Ref. 109
Journal code: FYB. ISSN: 0390-6078.
PUB. COUNTRY: Italy
Journal: Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, ACADEMIC)
LANGUAGE: English
ENTRY MONTH: 199512
AB Myelodysplastic syndromes (MDS) are clonal disorders of the multipotent
hematopoietic stem cell characterized by ineffective hematopoiesis and associated with marrow hypercellularity, increased intramedullary cell death and peripheral cytopenias of varying severity. Patients with myelodysplasia have a propensity (20% to 30% of cases) to undergo transformation into acute myeloid leukemia (AML), and a large body of evidence indicates that MDS represent steps in the multiphasic evolution of AML. Progression of the disease is characterized by expansion of the abnormal clone and **inhibition** of normal **hematopoiesis** leading to deterioration of the blood cell count and/or development of AML. MDS are relatively unusual in childhood, representing only 3% of pediatric hematological malignancies, although it has been reported that up to 17% of pediatric AML cases may have a previous myelodysplastic phase. The first systematic attempt at morphological classification of MDS
was provided by the French-American-British (FAB) group. However, the FAB
classification of MDS is only partially applicable in children. Some variants are extremely rare or absent (refractory anemia with ring sideroblasts and chronic myelomonocytic leukemia), and other peculiar pediatric disorders, represented by juvenile chronic myelogenous leukemia
(JCML) and the monosomy 7 syndrome, are not included. Moreover, since there is a partial overlap between pediatric MDS and myeloproliferative disorders and the variants occurring in young children have rather specific features, some confusion still surrounds the nosographical definition of childhood MDS, so that none of the proposed classifications are widely accepted and used. Characteristically, some genetic conditions such as Fanconi's anemia, Shwachman's and Down's syndromes predispose to
the development of MDS in childhood. The most common variants of childhood
MDS are represented by JCML and the monosomy 7 syndrome, both disorders
typically occurring in young children. JCML is characterized by a spontaneous growth of granulocyte-macrophage progenitors that show a striking hypersensitivity to granulocyte-macrophage colony-stimulating factor. Clinical presentation resembles that of some myeloproliferative disorders, with massive organomegaly usually not observed in the classically reported variants of MDS. Clinical features of the monosomy 7 syndrome resemble those observed in JCML and a differential diagnosis between these two entities relies upon the higher percentage of fetal **hemoglobin**, the more pronounced decrease in platelet count and, in some cases, the lack of the peculiar cytogenetic abnormality in the latter. With the number of children being cured of cancer constantly rising, a significant increase in secondary or chemotherapy-related myelodysplasia is being observed, and these disorders represent a formidable challenge for pediatric hematologists due to their poor response to chemotherapy.(ABSTRACT TRUNCATED AT 400 WORDS)

L9 ANSWER 10 OF 31 MEDLINE DUPLICATE 9
ACCESSION NUMBER: 95325339 MEDLINE
DOCUMENT NUMBER: 95325339
TITLE: Peripheral blood **stem cell** collection with **reduced** platelet loss to the patient/donor.
AUTHOR: Orlina A R; DeChristopher P J; Conant J C; Catchatourian R, Cano P
CORPORATE SOURCE: Department of Pathology, Michael Reese Hospital and Medical
Center, Chicago, IL 60616, USA..
SOURCE: JOURNAL OF CLINICAL APHERESIS, (1995) 10 (1) 1-6.
Journal code: HID. ISSN: 0733-2459.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199510
AB Apheresis procedures that optimize peripheral blood **stem cell** (PBSC) harvesting also result in a significant loss of platelets to the patient/donor because of their similar densities. We compared the percent
drop in platelet count and **hemoglobin** concentration in the patients before and after PBSC collection using two different collection chambers with the CS-3000. A modified plateletpheresis procedure was utilized. Seven patients underwent 38 PBSC collections during steady state
hematopoiesis using the standard A-35 collection chamber. At the end of the procedure, a second low-speed centrifugation of the PBSC concentrate
was performed in the manual mode, with siphoning out and return of the PRP
to the patient through a transfer pack. For 14 patients who underwent 113 PBSC collections, a small volume collection chamber (SVCC) was substituted
for the A-35 chamber and the second centrifugation step was omitted. These
patients were also primed with 4 g/m2 of cyclophosphamide. The percent drop in platelet count in the patients after the collection procedures was significantly less in the SVCC group (20.4 +/- 9.1 vs. 36.0 +/- 12.3, P = 0.000), even after correction for the difference in the volume of blood processed between the two groups (3.2 +/- 1.4 vs. 3.9 +/- 1.3, P = 0.006). The percent drop in **hemoglobin** concentration was also less with the SVCC both before (5.4 +/- 3.8 vs. 11.7 +/- 3.0, P = 0.000) and after (0.8 +/- 0.6 vs. 1.3 +/- 0.3, P = 0.000) correction for the difference in the volume of blood processed.(ABSTRACT TRUNCATED AT 250 WORDS)

L9 ANSWER 11 OF 31 MEDLINE DUPLICATE 10
ACCESSION NUMBER: 94191245 MEDLINE
DOCUMENT NUMBER: 94191245
TITLE: Basic fibroblast growth factor antagonizes transforming growth factor beta-mediated erythroid differentiation in K562 cells.
AUTHOR: Burger P E; Dowdle E B; Lukey P T; Wilson E L
CORPORATE SOURCE: Department of Clinical Science and Immunology, University
of Cape Town Medical School, South Africa.
CONTRACT NUMBER: CA49419 (NCI)
SOURCE: BLOOD, (1994 Apr 1) 83 (7) 1808-12.
Journal code: A8G. ISSN: 0006-4971.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals. Cancer
Journals
ENTRY MONTH: 199407
AB Basic fibroblast growth f[illegible] beta
1 (TGF-beta) have both be[illegible] cells. bFGF is a hematopoie[illegible] concert with other cytokin[illegible] induces erythroid differe[illegible] bFGF
might act on progenitor cel[illegible] that induce differentiatio[illegible] TGF-beta-mediated induct[illegible] cells. bFGF antagonized the[illegible] **hemoglobin** in a dose-deper[illegible] inhibiting **hemoglobin** induc[illegible] completely abrogating **hemo**[illegible] effective at antagonizing th[illegible] **hemoglobin** if it and TGF-be[illegible] cells, but delayed additio[illegible] resulted in significant inhit[illegible] inhibitory effects of bFGF[illegible] reversible, showing that bF[illegible] K562 cells. The hemin-medi[illegible] in K562 cells was only parti[illegible] expression of glycophorin A[illegible] indicate that bFGF might [illegible] numbers by **antagonizing** th[illegible] differentiation, thereby i[illegible] progenitor/stem cells.

L9 ANSWER 12 OF 31 MED[illegible] DUPLICA
ACCESSION NUMBER 940[illegible]
DOCUMENT NUMBER 940[illegible]
TITLE: Fetal hemato[illegible] transplantat[illegible] beta-thalassemic[illegible]
COMMENT: Comment [illegible] U.S.A 1[illegible] 1;90(21):10120-4
AUTHOR: Bethel C.A.[illegible] M.R.; M[illegible] E.R.
CORPORATE SOURCE: Dep[illegible] versit[illegible] San
Francisco
SOURCE: JOURNAL [illegible] EOLOGY, (19[illegible] 1232-7; discussion 1237-8
Journal code: JM[illegible]
PUB. COUNTRY: United S[illegible] (DUPLICATE PUB[illegible]
Journal; Article[illegible]
LANGUAGE: English
FILE SEGMENT: Priority
ENTRY MONTH: 199403
AB The transplantation of f[illegible] topoietic ste[illegible] may
potentially be used to tre[illegible] ties, immuno[illegible] storage diseases. The leve[illegible] engraftment [illegible] reconstitute the recipient[illegible] system are d[illegible] and remain unknown for m[illegible] have exp[illegible] application
of fetal hematopoietic ste[illegible] for the ame[illegible] hemolytic disease in a mur[illegible] thalassemic [illegible] neonatal homozygous beta [illegible] were transf[illegible] intraperitoneally (IP) with[illegible] cells from syn[illegible] nonthalassemic murine fet[illegible] sis gestat[illegible] **hemoglobin** was demonstr[illegible] blood of[illegible] transplant recipients at le[illegible] 44% to 27[illegible]

levels of engraftment in 6 of these 9 transplant chimeras remained stable or increased up to 150 days after transplantation, with levels ranging from 13.6% to 54.6% at 280 days. Three chimeras have demonstrated gradually decreasing engraftment after 200 days. The degree of engraftment correlated with clinically relevant improvement: decreased reticulocyte counts (8.4% to 15.7% in chimeras [n = 9] v 17.1% to 19.1% in controls [n = 8], P = .01), increased mean RBC deformability, and the significant **reduction** in extramedullary **hematopoiesis** and iron deposits seen on histological examination of chimeric liver and spleen. These data demonstrate that fetal HSC transplants results in significant long-term chimerism with favorable alterations in red cell characteristics, and decreased hemolytic anemia in beta-thalassemia.

L9 ANSWER 13 OF 31 MEDLINE DUPLICATE 12
ACCESSION NUMBER: 93375581 MEDLINE
DOCUMENT NUMBER: 93375581
TITLE: Detection of erythropoietic inhibitor factor in a case of acute non-lymphocytic leukemia after allogenic bone marrow transplantation.
AUTHOR: Kurosawa K; Aizawa S; Hojo H; Kawanishi Y; Kimura Y; Toyama K
CORPORATE SOURCE: First Department of Internal Medicine, Tokyo Medical College..
SOURCE: RINSHO KETSUEKI. JAPANESE JOURNAL OF CLINICAL HEMATOLOGY, (1993 Jun) 34 (6) 748-52.
Journal code: KII. ISSN: 0485-1439.
PUB. COUNTRY: Japan
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: Japanese
ENTRY MONTH: 199312
AB A 36-year-old man was diagnosed as having acute non-lymphocytic leukemia (AML M5b) in 1985 and received allogenic bone marrow transplantation from an ABO-mismatched sibling in January 1987. Recovery of erythropoiesis in this patient was delayed, then the **hemoglobin** level improved in parallel with disappearance of anti-A antibody in the serum on day 260 post transplantation. However, as anemia occurred again despite no [redacted]ipse of leukemia on day 350, we tried to determine the presence of erythropoietic inhibitory factor in this patients. Erythroid colony formation was decreased when bone marrow cells were co-cultured with peripheral mononuclear cells from the patient. Further, erythroid colony inhibitory activity was found in conditioned medium of PHA-stimulated T cells from the patient. Sephadex gel fractionation showed that the molecular weight of this inhibitory factor was approximately 11,000 and addition of a high concentration of EPO did not eliminate the inhibitory activity. These findings suggest that the novel inhibitor described in this manuscript, produced by T cells, was differed from previously reported inhibitors such as anti-EPO antibody, spermine and uremic toxins.

L9 ANSWER 14 OF 31 MEDLINE DUPLICATE 13
ACCESSION NUMBER: 94003392 MEDLINE
DOCUMENT NUMBER: 94003392
TITLE: Tumor **inhibition** and **hematopoietic** stimulation in mice by a synthetic copper-ATP complex.
AUTHOR: Pal S; Ray M R; Maity P
CORPORATE SOURCE: Department of Cell Biology, Chittaranjan National Cancer Institute, Calcutta, India..
SOURCE: ANTI-CANCER DRUGS, (1993 Aug) 4 (4) 505-10.
Journal code: A9F. ISSN: 0959-4973.
PUB. COUNTRY: ENGLAND, United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199401
AB The hematologic effect of [Cu3(ATP)(2)6H2O]2-, a synthetic copper-ATP complex (Cu-ATP) having antitumor activity, was investigated in normal and Ehrlich ascites carcinoma-bearing mice. Cu-ATP (25 mg/kg) induced appreciable tumor inhibition and prolonged host survival which were accompanied by elevated levels of **hemoglobin**, platelet and lymphocytes while total WBC count and bone marrow cellularity remained unaffected. In normal mice the compound elicited marrow and splenic hypercellularity with a greater number of granulocyte progenitors and elevated levels of peripheral WBC, RBC and platelets. In addition, the total number of CFU-S of these treated animals was increased and these pluripotent stem cells differentiate preferentially towards granulocyte lineage. The results indicate that Cu-ATP does not adversely affect **hematopoiesis** while it **inhibits** tumor growth; on the contrary, it has a stimulatory effect on murine granulocytopoiesis.

L9 ANSWER 15 OF 31 MEDLINE DUPLICATE 14
ACCESSION NUMBER: 93114386 MEDLINE
DOCUMENT NUMBER: 93114386
TITLE: The effect of nordihydroguaiaretic acid, an inhibitor of prostaglandin and leukotriene biosynthesis, on hematopoiesis of gamma-irradiated mice [published erratum appears in Exp Hematol 1993 Apr;21(4):593].
AUTHOR: Kozubik A, Hofmanova J, Hola J, Netikova J
CORPORATE SOURCE: Institute of Biophysics, Czechoslovak Academy of Sciences, Brno..
SOURCE: EXPERIMENTAL HEMATOLOGY, (1993 Jan) 21 (1) 138-42.
Journal code: EPR. ISSN: 0301-472X
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Cancer Journals; Priority Journals
ENTRY MONTH: 199304
AB The effects of the inhibition of the cyclooxygenase and lipoxygenase metabolic pathways of arachidonic acid on the postirradiation recovery of hematopoietic functions in mice were investigated. Nordihydroguaiaretic acid (NDGA), an inhibitor of prostaglandin (PG) and leukotriene (LT) production, was given to animals in single doses (0.015 to 0.75 mg/mouse) 1 hour before 5 Gy of total-body gamma-irradiation. Enhanced hematopoietic recovery in terms of exogenous and endogenous spleen colonies, femoral granulocyte-macrophage colony-forming cells and peripheral blood granulocyte levels was observed at higher doses of NDGA. The treatment used influenced neither lymphocyte nor erythrocyte postirradiational levels or **hemoglobin** concentration. A comparison of the effects induced by a high dose of NDGA (0.3 mg per mouse) with those observed after an isomolar dose of indomethacin (an inhibitor of PG production) indicated only slight differences between these two drugs. An isomolar dose of esculetin (an inhibitor of LT production) had no effect on the postirradiation behavior of **hematopoiesis**. The results suggest that the **inhibition** of PG production plays the main role in the mechanism of NDGA action. Inhibition of LT production seems to be of less importance for hematopoiesis in these in vivo conditions.

L9 ANSWER 16 OF 31 MED[redacted]
ACCESSION NUMBER: 912[redacted]
DOCUMENT NUMBER: 912[redacted]
TITLE: Negative corr[redacted] neopterin concentr[redacted]
AUTHOR: Fuchs D; Sh[redacted] Reibnegger G; We[redacted]
CORPORATE SOURCE: Inst[redacted] University of Innsbruck, Aus[redacted]
SOURCE: AIDS, (1991[redacted] Journal code: AI[redacted]
PUB. COUNTRY: United S[redacted] Journal; Article; [redacted]
LANGUAGE: English
FILE SEGMENT: Priority
ENTRY MONTH: 199108
AB Hematopoietic disturban[redacted] infection. Recent studies on immune [redacted] demonstrate that HIV-1 infection is ass[redacted] investigated a possible ass[redacted] concentrations and blood cell counts (CD4+[redacted] blood cells) and **hemoglobin**[redacted] HIV-1-seropositive individ[redacted] one WR5, and 10 WR6]. There [redacted] neopterin concentrations o[redacted] hematocrit and platelets. [redacted] either only WR1 and WR2 [redacted] considered for calculations. Thus, hem[redacted] chronic immune activation [redacted] of neopterin are released [redacted] interferon-gamma (IFN ga[redacted] alpha) further enhances the effe[redacted] that activated immune cells and[redacted] TNF alpha are involved **inhibiting**[redacted]

L9 ANSWER 17 OF 31 CAN[redacted]
ACCESSION NUMBER: 916[redacted]
DOCUMENT NUMBER: 916[redacted]
TITLE: [STUDY OF G[redacted] TRANSFORMING GROWTH FACTOR IN ACU[redacted] LYMPHOPROLIFERATIVE SYNDROMES]. ETUDE DE LA TR[redacted] TRANSFORMING GROWTH FACTOR BETA D[redacted] SYNDROMES LYMPHOPROLIF[redacted]
AUTHOR: Le Rohellec [redacted]
CORPORATE SOURCE: Unive[redacted]
SOURCE: Diss Abstr [redacted] ISSN: 0419-4217
DOCUMENT TYPE: (THE[redacted]
FILE SEGMENT: ICDB[redacted]

LANGUAGE: French
ENTRY MONTH: 199104
AB Transforming growth factor beta 1 (TGF beta 1) is a polypeptide factor that stimulates or inhibits the proliferation and differentiation of cells in vitro, depending on the type of cell and the factors present in the medium. Since the sum of these effects on **hematopoietic** cells is essentially **inhibitory**, TGF beta 1 could mediate the **inhibition** of normal **hematopoiesis** seen in certain hemopathies. We have studied the transcription of the TGF beta 1 gene in the promyelocyte line HL-60 and in 50 malignant hemopathies: 16 nonlymphoblastic acute leukemias 11 acute myelodysplastic syndromes, 11 myeloproliferative syndromes; 6 acute lymphoblastic leukemias, and 6 lymphoproliferative conditions. One sign was observed in HL-60 and in most of the hemopathies studied. The level of transcription was very variable between diseases. By simultaneous study of the glyceraldehyde 3 phosphate [illegible]dehydrogenase gene as control, it was possible to compare the level of [illegible]transcription of the TGF beta 1 gene in 30 hemopathies and refer it to the blood count of the time of the study. Transcription of TGF beta 1 is significantly greater in the acute myeloproliferative syndromes than in the other dyscrasias. An elevated level of transcription is associated with a thrombopenia below 100,000/mm3. No significant correlation was observed between level of transcription and **hemoglobin** titer or the rate of blast cell proliferation at the time of study. Further studies are needed to determine if this transcription is accompanied by secretion of TGF beta 1 by malignant cells and in what degree the growth factor participates in the regulation of blast proliferation or in the **inhibition** of normal **hematopoiesis**.

L9 ANSWER 18 OF 31 MEDLINE DUPLICATE 16
ACCESSION NUMBER: 89321405 MEDLINE
DOCUMENT NUMBER: 89321405
TITLE: Primary myelofibrosis-osteomyelosclerosis (agnogenic myeloid metaplasia): correlation of clinical findings with bone marrow histopathology and prognosis.
AUTHOR: Thiele J; Steinberg T; Zankovich R; Fischer R
CORPORATE SOURCE: Institute of Pathology, University of Cologne, F.R.G..
SOURCE: ANTICANCER RESEARCH, (1989 Mar-Apr) 9 (2) 429-35.
Journal code: 59L. ISSN: 0250-7005.
PUB. COUNTRY: Greece
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; Cancer Journals
ENTRY MONTH: 198910
AB A clinicopathological study was performed on 90 patients (39 males - 51 females, age 68 years) with primary (idiopathic) myelofibrosis - osteomyelosclerosis (OMF) in order to correlate laboratory and histomorphological parameters with each other and to calculate factors of prognostic impact on survival. In addition to multiple interactions between various laboratory features, there was a significant correlation between degree of medullary fibrosis and osteosclerotic changes with sizes of spleen and liver, level of LDH and duration of relevant prediagnostic symptoms. In trephine biopsies of the bone marrow, **reduction** of **hematopoietic** tissue was assessed by evaluating the amount of fat cells plus the degree of osteosclerotic lesions. This histological parameter did not reveal significant relationships with hepatosplenomegaly, duration of relevant symptoms or length of disease, but was correlated with the clinical findings of bone marrow failure. On univariate analysis, several clinical (age greater than 45 years, presence of relevant prediagnostic symptoms, **hemoglobin** level less than 9 g/dl, counts of myelo- and normoblasts, thrombocyte count less than 100 and greater than 700 x 10(9)/l, spleen size and LDH level) and histological features (**reduction** of **hematopoiesis**, counts for megakaryocytes and lymphoid nodules) were found to exert a predictive value on prognosis. However, on multivariate regression analysis only age remained significant. This result apparently reflects the numerous interactions between the various clinical as well as histological variables tested.

L9 ANSWER 19 OF 31 MEDLINE DUPLICATE 17
ACCESSION NUMBER: 89232049 MEDLINE
DOCUMENT NUMBER: 89232049
TITLE: Transferrin receptor-mediated suppression of in vitro hematopoiesis by transferrin-gallium.
AUTHOR: Chitambar C R; Craig A; Ash R C
CORPORATE SOURCE: Department of Medicine, Medical College of Wisconsin, Milwaukee 53226.
CONTRACT NUMBER: CA41740 (NCI)
SOURCE: EXPERIMENTAL HEMATOLOGY, (1989 Jun) 17 (5) 418-22.
Journal code: EPR. ISSN: 0301-472X.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; Cancer Journals
ENTRY MONTH: 198908
AB The expression of transferrin receptors on cells is felt to reflect iron requirements for proliferation or for **hemoglobin** production. We have recently shown that transferrin-gallium (Tf-Ga) complexes bind to cellular transferrin receptors and inhibit cellular iron incorporation. In this study, Tf-Ga in a dose-dependent manner inhibited the growth of erythroid (erythroid burst-forming units [BFU-E]-derived), granulocyte-macrophage (granulocyte-macrophage colony-forming units [CFU-GM]-derived) and mixed (mixed CFU [CFU-GEMM]-derived) hematopoietic colonies. Although major differences in the response of the different progenitor cells to Tf-Ga were not seen, CFU-GEMM-derived colonies appeared to be more sensitive to growth inhibition by Tf-Ga. The inhibitory effects on colony growth were reversible after 48 h of exposure of marrow cells to Tf-Ga, suggesting that the initial effects of Tf-Ga were mainly cytostatic and that continuous exposure of cells to Tf-Ga was required for maximal growth inhibition. Transferrin-iron (Tf-Fe) added to the Tf-Ga-containing cultures restored colony growth; however, this effect was best seen when Tf-Fe was added at day 0 of incubation. Tf-Fe added on days 3 or 7 failed to restore GEMM colonies and restored only a fraction of BFU-E and GM colonies. Tf-Ga appears to **inhibit** **hematopoietic progenitor cell** growth by interfering with cellular iron utilization during an early phase of progenitor cell proliferation. The use of Tf-Ga may allow further exploration of the role of iron and the Tf receptor in the regulation of hematopoietic progenitor cell growth.

L9 ANSWER 20 OF 31 BIOSIS COPYRIGHT 1999 BIOSIS
ACCESSION NUMBER: 1989:52818 BIOSIS
DOCUMENT NUMBER: BA87:28818
TITLE: DOPAMINE INHIBITS TUMOR GROWTH AND CONCOMITANTLY STIMULATES ERYTHROCYTE AND PLATELET PRODUCTION IN EXPERIMENTAL TUMOR BEARING MICE.
AUTHOR(S): RAY M R; DASGUPTA P S; BASU S; LAHIRI T
CORPORATE SOURCE: DEP[illegible] AKAN[illegible]
CANCER RES. CENT., 37 S.P. MOOKERJEE [illegible]
SOURCE: BIOG AMIN[illegible] CODEN: BIAME7
FILE SEGMENT: BA; OLD
LANGUAGE: English
AB Effects of dopamine (DA [illegible]tumor[illegible] nematological abnormalities [illegible]strain A mice [illegible] transplantable Ehrlich ascit[illegible] A was administer[illegible] intraperitoneally on day 1 ar[illegible] transplan[illegible] of 50 mg/kg per day for 7 [illegible] mice [illegible] similarly. The animals were [illegible] completion [illegible] treatment schedule. The treatment co[illegible] [illegible]tion of [illegible] tumor cell count, and an appr[illegible] the host [illegible] Tumor inhibition was accom[illegible] improvement [illegible] **hemoglobin** concentration, [illegible] marrow ce[illegible] and stimulated production [illegible] and platelets [illegible] normal mice also elicited en[illegible] [illegible]tein concentrat[illegible] RBC counts, peripheral grar[illegible] [illegible] reused bone [illegible] count, splenomegaly, and st[illegible] [illegible]esis and the [illegible] The results suggest that DA do[illegible] [illegible]ffect **hematop**[illegible] while it **inhibits** tumor grow[illegible] [illegible] has stimulatory effect one en[illegible] [illegible]rtially ame[illegible] anemia that accompanies t[illegible]

L9 ANSWER 21 OF 31 BIO[illegible] [illegible]1999 BIOSI[illegible]
18
ACCESSION NUMBER: 198[illegible]
DOCUMENT NUMBER: BA8[illegible]
TITLE: [illegible]ID PROGENI[illegible] REDUCED IN [illegible] CELL GROWTH IN[illegible] ANCER
AUTHOR(S): MASTERS [illegible] BAILEY-WO[illegible] T; LITTLEWOOD T; BENTLEY P; PA[illegible] JACOBS A
CORPORATE SOURCE: DEP[illegible] [illegible]GP, W.A[illegible] PARK, CARDIFF CF4 4XW.
SOURCE: J CLIN PAT[illegible] [illegible]7) 40 (1), 8[illegible] CODEN: JCPAAK
FILE SEGMENT: BA; OLI
LANGUAGE: English
AB Peripheral blood and bon[illegible] [illegible]ied in 21 [illegible] disseminated [illegible] untreated bronchial cance[illegible] [illegible] define abno[illegible] erythropoiesis associated [illegible] [illegible]ment of anaem[illegible] concentration at or below [illegible]nt in 13 cases. N[illegible] morphology was normal in [illegible]e, in which s[illegible] tumour cells were found. C[illegible] [illegible]s, thyroid proge[illegible] significant decrease in the [illegible] ($p = 0.03$) [illegible] 0.01) compared with cultur[illegible] marrow (12 s[illegible] growth of granulocyte and macro[illegible] (GM-CFCs) w[illegible] patients with bronchial ca[illegible] subjects. Whe[illegible] was incubated in the presence [illegible] normal cancer[illegible] inhibitory factors could b[illegible] for BFU-E[illegible] In all patients circulating TB[illegible] [illegible]ificantly re[illegible] 0.0002). Consequently, the[illegible] [illegible]etic in blood w[illegible] was significantly lower th[illegible] found in 2[illegible] ($p = 0.036$). In 18 patients[illegible] [illegible]4 TB pati[illegible]

significantly lower than the ratio of 2.9 found in seven normal subjects (p = 0.04). Total blood white cell counts, neutrophils, and monocyte numbers were also increased (p = 0.0001; p = 0.0001; p = 0.002).

L9 ANSWER 22 OF 31 MEDLINE DUPLICATE 19
ACCESSION NUMBER: 84128903 MEDLINE
DOCUMENT NUMBER: 84128903
TITLE: Effect of age on hematopoiesis in man.
AUTHOR: Lipschitz D.A; Udupa K.B; Milton K.Y; Thompson C.O
CONTRACT NUMBER: AG02603 (NIA)
SOURCE: BLOOD, (1984 Mar) 63 (3) 502-9.
Journal code: ABG. ISSN: 0006-4971.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals; Cancer
Journals
ENTRY MONTH: 198406
AB We have shown previously that the cause of anemia in healthy elderly subjects can usually not be identified. In this study, hematopoiesis was examined in 18 healthy elderly subjects with unexplained anemia and in 15 young and 15 healthy elderly individuals without anemia. No reduction in circulating testosterone was noted, making decreased androgen levels as a cause for the anemia unlikely. The 2,3 diphospho-glycerate (2,3DPG) levels in the anemic subjects were significantly higher than their corresponding controls, suggesting that the anemia was pathologic, as no increase would be expected if the low **hemoglobin** was a physiologic adjustment to age. The anemia was associated with a reduction in marrow normoblast and CFU-E number, but no decrease in BFU-E levels was seen. This suggests that the mechanism of the anemia is a decrease in stem cell proliferation. This could be caused by a reduction in circulating erythropoietin or a defect in end organ response. A second possibility is that a basic cellular abnormality exists. The presence of an overall **reduction** in **hematopoiesis** in anemic elderly (decreased peripheral blood counts, reduced marrow myeloid precursors, and CFU-C levels) makes this especially likely. The abnormality may be caused by a mechanism unrelated to the aging process. The fact that nonanemic elderly also have **reductions** in **hematopoiesis** suggests that age contributes to the defect.

L9 ANSWER 23 OF 31 MEDLINE
ACCESSION NUMBER: 84080823 MEDLINE
DOCUMENT NUMBER: 84080823
TITLE: Perturbations in the erythroid marrow progenitor cell pools may play a role in the augmentation of HbF by 5-azacytidine.
AUTHOR: Torrealba-de Ron A.T; Papayannopoulou T; Knapp M.S; Fu M.F; Knitter G; Stamatoyannopoulos G
CONTRACT NUMBER: HL 20899 (NHLBI)
RR 00166 (NCRR)
SOURCE: BLOOD, (1984 Jan) 63 (1) 201-10.
Journal code: ABG. ISSN: 0006-4971.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals; Cancer
Journals
ENTRY MONTH: 198404
AB In vivo observations on the kinetics of F cells and of fetal **hemoglobin** (HbF) synthesis and in vitro studies of erythroid progenitors, their number, and the gamma-gene expression in their progeny were carried out in baboons (Papio cynocephalus) treated with 5-azacytidine. Maximum effect on the increase of HbF production in vivo was observed only when an expanded erythroid marrow population was present. In these animals, as well as in normal animals, treatment resulted in a significant **reduction** of the late erythroid **progenitor cell** pools (erythroid clusters and erythroid colony-forming units, CFU-E) in the marrow. This reduction was more pronounced among those progenitors grown in the absence of added erythropoietin, and it was followed by a rebound a few days after treatment cessation, reflecting the accumulation of regenerating progenitors. An early increase in the in vitro synthesis of HbF in erythroid clusters and CFU-E colonies was observed. This increase was further documented at the cellular level, with immunofluorescent labeling of colonies with monoclonal anti-gamma-globin chain antibodies. In contrast to the findings in late progenitors, the number of erythroid burst-forming unit (BFU-E) colonies and the synthesis of HbF in these colonies was not influenced significantly by 5-azacytidine treatment. It is proposed that the toxic effects of 5-azacytidine on late progenitors, leading to faster mobilization of earlier progenitors to the next more mature compartment, play a role in the in vivo augmentation of HbF synthesis by this drug. This perturbation in the progenitor cell population kinetics and the presumed hypomethylation of the surviving differentiating cells may act synergistically to produce a maximum HbF response after 5-azacytidine treatment.

L9 ANSWER 24 OF 31 EMBASE COPYRIGHT 1999 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 84049488 EMBASE
DOCUMENT NUMBER: 1984049488
TITLE: Perturbations in the erythroid marrow progenitor cell pools may play a role in the augmentation of HbF by 5-azacytidine.
AUTHOR: Torrealba de Ron A.T.; Papayannopoulou T.; Knapp M.S.; et al.
CORPORATE SOURCE: Department of Medicine, RG-25, University of Washington, Seattle, WA 98195, United States
SOURCE: Blood, (1984) 63/1 (201-210).
CODEN: BLOOAW
COUNTRY: United States
DOCUMENT TYPE: Journal
FILE SEGMENT: 037 Drug Literature Index
025 Hematology
022 Human Genetics
030 Pharmacology
LANGUAGE: English
AB In vivo observations on the kinetics of F cells and of fetal **hemoglobin** (HbF) synthesis and in vitro studies of erythroid progenitors, their number, and the .gamma.-gene expression in their progeny were carried out in baboons (Papio cynocephalus) treated with 5-azacytidine. Maximum effect on the increase of HbF production in vivo was observed only when an expanded erythroid marrow population was present. In these animals, as well as in normal animals, treatment resulted in a significant **reduction** of the late erythroid **progenitor cell** pools (erythroid clusters and erythroid colony-forming units, CFU-E) in the marrow. This reduction was more pronounced among those progenitors grown in the absence of added erythropoietin, and it was followed by a rebound a few days after treatment cessation, reflecting the accumulation of regenerating progenitors. An early increase in the in vitro synthesis of HbF in erythroid clusters and CFU-E colonies was observed. This increase was further documented at the cellular level, with immunofluorescent labeling of colonies with monoclonal [illegible] contrast to the findings in [illegible] burst forming unit (BFU-E) [illegible] colonies was not influenced [illegible] is proposed that the toxic [illegible] leading to faster mobilizat[illegible] mature compartment, play a [illegible] synthesis by this drug. This [illegible] population kinetics and the [illegible] differentiating cells may ac[illegible] response after 5-azacytidin[illegible]

L9 ANSWER 25 OF 31 MEDL[illegible]
ACCESSION NUMBER: 8330[illegible]
DOCUMENT NUMBER: 8330[illegible]
TITLE: Direct evidenc[illegible] between human progenitor cells an[illegible] activity present in[illegible]
AUTHOR: Stamatoyann[illegible] Papayannopoulou T[illegible]
CONTRACT NUMBER: HL 20[illegible] GM 15253 (NIGM[illegible] RR-37 (NCRR)
SOURCE: PROCEEDIN[illegible] NAL ACAD[illegible] SCIENCES OF THE UNITED STATES[illegible] Journal code: PV3[illegible]
PUB. COUNTRY: United St[illegible] Journal; Article; ([illegible]
LANGUAGE: English
FILE SEGMENT: Priority [illegible]
ENTRY MONTH: 198312
AB An activity that induces [illegible] in fetal sheep serum. To tes[illegible] interactions in **hemoglobin** s[illegible] erythroid cell differentiat[illegible] transfer experiments were [illegible] switching activity-containing medium [illegible] calf serum) were transferr[illegible] sheep serum or fetal calf s[illegible] determined in the fully mature erythro[illegible] transfers produced colonie[illegible] undisturbed fetal calf seru[illegible] calf serum transfers resulted [illegible] revealing an interaction be[illegible] and cells at an early stage [illegible] development. The **reduction** serum-to-fetal sheep serum switching activity interacts development. Maximal decr[illegible] sheep serum-to-fetal shee[illegible] effects on Hb switching ar[illegible] Hb switching is present throu[illegible] splitting single early clones either a fetal sheep serum interactions were further [illegible] erythroid burst-forming u[illegible] cell-free plates, the result[illegible] of fetal sheep serum-to-fe[illegible] switching activity does no[illegible]

studies show directly that (i) Hb F synthesis is controlled at the level of progenitors and (ii) it involves interactions between progenitor cells and their environment.

L9 ANSWER 26 OF 31 EMBASE COPYRIGHT 1999 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 84024833 EMBASE
DOCUMENT NUMBER: 1984024833
TITLE: Direct evidence for interaction between human erythroid progenitor cells and a **hemoglobin** switching activity present in fetal sheep serum.
AUTHOR: Stamatoyannopoulos G.; Nakamoto B.; Kurachi S.; Papayannopoulou T.
CORPORATE SOURCE: Division of Medical Genetics, University of Washington, Seattle, WA 98195, United States
SOURCE: Proceedings of the National Academy of Sciences of the United States of America, (1983) 80/181 (5650-5654). CODEN: PNASA6
COUNTRY: United States
DOCUMENT TYPE: Journal
FILE SEGMENT: 021 Developmental Biology and Teratology
LANGUAGE: English
AB An activity that induces Hb F to Hb A switching in human cells is present in fetal sheep serum. To test directly the role of cell-to-environment interactions in **hemoglobin** switching and to define the level of erythroid cell differentiation at which this activity operates, colony transfer experiments were done. Clones grown in the presence of switching activity-containing medium (fetal sheep serum) or control medium (fetal calf serum) were transferred, at the 16- to 30-cell stage, to either fetal sheep serum or fetal calf serum plates and Hb F synthesis was determined in the fully mature erythroid bursts. Fetal calf serum-to-fetal calf serum transfers produced colonies with the high Hb F levels characteristic of undisturbed fetal calf serum-grown clones. Fetal sheep serum-to-fetal calf serum transfers resulted in significant decrease in Hb F synthesis, revealing an interaction between **hemoglobin** switching activity and cells at an early stage of **progenitor cell** development. The **reduction** of Hb F synthesis in fetal calf serum-to-fetal sheep serum transfers indicated that **hemoglobin** switching activity interacts with cells at later stages of progenitor cell development. Maximal decrease in Hb F synthesis was observed in fetal sheep serum-to-fetal sheep serum transfers, indicating that optimal effects on Hb switching are obtained when the environment that induces Hb switching is present throughout the development of progenitor cells. By splitting single early clones into two parts and transferring them to either a fetal sheep serum or a fetal calf serum environment, these interactions were further demonstrated in the progeny of a single erythroid burst-forming unit. Since all clone transfers were done on cell-free plates, the results of fetal calf serum-to-fetal sheep serum and of fetal sheep serum-to-fetal sheep serum transfers indicated that the switching activity does not require helper cells for its action. These studies show directly that (i) Hb F synthesis is controlled at the level of progenitors and (ii) it involves interactions between progenitor cells and their environment.

L9 ANSWER 27 OF 31 MEDLINE DUPLICATE 20
ACCESSION NUMBER: 83239504 MEDLINE
DOCUMENT NUMBER: 83239504
TITLE: Iron status and anemia in the elderly: new findings and a review of previous studies.
AUTHOR: Garry P.J.; Goodwin J.S.; Hunt W.C.
CONTRACT NUMBER: AG02049 (NIA)
RR-00997-05,06 (NCRR)
SOURCE: JOURNAL OF THE AMERICAN GERIATRICS SOCIETY, (1983 Jul) 31 (7) 389-99.
Journal code: H6V. ISSN: 0002-8614.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198310
AB Iron status was determined in 280 free-living and healthy elderly men (n = 131) and women (n = 149) by assessing dietary and supplemental iron intake as well as ten biochemical measures of iron nutriture (erythrocyte count, **hemoglobin** level, hematocrit, mean corpuscular volume, mean corpuscular **hemoglobin**, mean corpuscular **hemoglobin** concentration, plasma iron level, total iron-binding capacity, per cent transferrin saturation, and ferritin level). Subject ages ranged from 60 to 93 years with a median age of 72 years for both women and men. For comparison purposes, iron status measures in an unselected group of younger men (n = 107) and women (n = 164) between the ages of 20 and 39 years were also obtained. None of the elderly women and only two (1.2 per cent) of the younger women had low **hemoglobin** levels (less than 12.0 g/dl). Three (2.3 per cent) of the elderly men and none of the younger men had low **hemoglobin** levels (less than 14 g/dl). Other iron status measures revealed that anemia or iron deficiency was no more prevalent in the healthy elderly population than in the younger adult population when identical criteria were used to assess iron nutriture. The genesis of anemia often seen in the elderly is not completely understood. Reported evidence suggests the presence of anemia in the elderly is a result of overall **reduction** of **hematopoietic** reserves. Because of the potentially serious consequences of this assumption about anemia to the treatment of the elderly, the authors critically review some of the studies that have been designed in the past to determine the prevalence and etiology of anemia in the aged. They suggest that health status, race, socioeconomic status, diet, and region are more important than age as explanations for the high prevalence of anemia seen in many previous studies.

L9 ANSWER 28 OF 31 EMBASE COPYRIGHT 1999 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 81024538 EMBASE
DOCUMENT NUMBER: 1981024538
TITLE: Chronic toxicity of aclacinomycin A in dogs.
AUTHOR: Kawamura K.; Tomizawa S.; Sato H.; et al.
CORPORATE SOURCE: Dept. Pharmacol., Sch. Pharmaceut. Sci., Kitasato Univ., Tokyo, Japan
SOURCE: Pharmacometrics, (1980) 19/5 (765-781). CODEN: OYYAA2
COUNTRY: Japan
DOCUMENT TYPE: Journal
FILE SEGMENT: 037 Drug Literature Index
016 Cancer
030 Pharmacology
LANGUAGE: Japanese
SUMMARY LANGUAGE: English
AB Male beagle dogs were treated with Aclacinomycin A (0.3, 0.6 or 0.9 mg/kg, i.v.), a new antitumor antibiotic, once a day for 10 months. The results obtained were as follows: 1. One dog given a dose of 0.9 mg/kg died. All other dogs survived. 2. Occasional vomiting and depression of spontaneous activity during the early stage of the administration period, and a decrease in body weight a[illegible] The hematologic values revealed [illegible] hematocrit and **hemoglobin** [illegible] were slightly decreased in [illegible] slightly decreased, and a [illegible] mg/kg groups. 5. Sites of [illegible] induration. 6. The histologic [illegible] atrophy of the testicles, re[illegible] **hematopoiesis** in the bone [illegible] groups. Hyperplasia and ne[illegible] tissue of injection sites. The mag[illegible] In the electron microscopic e[illegible] electron density of the mit[illegible] mg/kg groups. Essentially [illegible] urinalysis, examination of [illegible]

L9 ANSWER 29 OF 31 ME[illegible] DUPLICA[illegible]
ACCESSION NUMBER: 791[illegible]
DOCUMENT NUMBER: 791[illegible]
TITLE: Evidence for [illegible] agent in [illegible] cyclic hematopoie[illegible] **hemoglobin** synthe[illegible]
AUTHOR: White J.F.; [illegible]
SOURCE: EXPERIEN[illegible] Journal code: E[illegible]
PUB. COUNTRY: Switzer[illegible] Journal; Article; [illegible]
LANGUAGE: English
FILE SEGMENT: Priority[illegible]
ENTRY MONTH: 19790[illegible]
AB Serum samples collected [illegible] of a cyclic **hematopoietic** (CH) dog u[illegible] spheric conditions, were assayed [illegible] affect **hemoglobin** synthesis by normal canine [illegible] ing levels [illegible] **hemoglobin** synthesis in th[illegible] ferent serum suggest an agent cycles in [illegible] ells which [illegible] **hemoglobin** synthesis.

L9 ANSWER 30 OF 31 EMB[illegible] 1999 ELSE[illegible]
ACCESSION NUMBER: 784[illegible]
DOCUMENT NUMBER: 197[illegible]
TITLE: The effect o[illegible] hemolysis [illegible] defense mechan[illegible]
AUTHOR: Gelman B.B.; [illegible] Bus J.S.
CORPORATE SOURCE: Div[illegible] Environm. Hlth [illegible] Coll. Med., Cincinnat[illegible] ted States
SOURCE: Toxicology [illegible] xicology, (19[illegible]
CODEN: TXAPA
COUNTRY: United St[illegible]
DOCUMENT TYPE: Journ[illegible]
FILE SEGMENT: 037 [illegible] dex
030 Pharmac[illegible]
025 Hematol[illegible]
LANGUAGE: English
AB The effect of chronic [illegible] ditive hem[illegible] biochemical defense mec[illegible] hemolysis [illegible] The interaction between lead [illegible] nazone (Ph[illegible] using four groups of adult male [illegible] id acetat[illegible]

oral-ip dosage regimen. After 27 days of expusure the blood lead (PbB) concentrations were (mean .+-. SD) 2.3 .+- 1 (control), 33 .+-. 4, 67 .+-. 13, and 104 .+-. 17 .mu.g/100 ml. On Day 27, PHZ (45 mg/kg sc) was administered to half of the rats in each group, and **hemoglobin** (Hb) and hematocrit (Hct) determinations were performed on tail blood drawn on Days 28, 29, 34, and 40. The results showed that in the acute hemolytic phase after PHZ both lead alone and PHZ alone reduced Hb and Hct but that the lead-PHZ interaction was not synergistic. A synergistic interaction did occur during the compensatory phase of anemia. The effect of in vitro lead exposure on in vitro hemolysis and biochemical defense mechanisms was studied in a second experiment, the results of which showed that lead caused a dose-dependent increase in oxidative **hemolysis** in vitro. Superoxide dismutase activity was decreased, whereas pentose shunt activity was increased. The effect of lead on reduced glutathione concentrations and glutathione peroxidase activity was biphsic, being increased at the intermediate dose but returning to baseline at the highest dose. It is concluded that the in vivo interaction between Pb concentrations of up to approximately 100 .mu.g/100 ml blood and oxidative hemolytic anemia was due to the ability of lead to **inhibit** compensatory **hematopoiesis** after an acute hemolytic episode. The more sensitive in vitro hemolysis test showed that lead caused a dose-dependent increase in oxidative hemolysis, and the biochemical changes observed were consistent with the hypothesis that in vivo lead exposure exerts a moderate pro-oxidant effect on rat erythrocytes.

L9 ANSWER 31 OF 31 MEDLINE DUPLICATE 22
ACCESSION NUMBER: 78007357 MEDLINE
DOCUMENT NUMBER: 78007357
TITLE: Anemia of lead intoxication: a role for copper.
AUTHOR: Klauder D S; Petering H G
SOURCE: JOURNAL OF NUTRITION, (1977 Oct) 107 (10) 1779-85.
Journal code: JEV. ISSN: 0022-3166.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197801
AB Lead-induced anemia in rats, which is of a microcytic, hypochromic type, has been shown to be a result of an interference with the metabolism of copper and iron. In this complex interaction, copper may be the target upon which ingested lead has its **antagonistic** effect on **hematopoiesis**. The depressions in hematocrit and **hemoglobin** levels resulting from exposure to lead may occur secondarily to the effects of a lead-induced copper deficiency on iron mobilization and utilization. The metabolic fault induced by lead is seen in a reduction of serum iron, elevation of serum iron binding capacity, and increase in liver iron, all manifestations of systemic effects related to an interference with copper metabolism. These results relate many of the characteristics of the lead-induced anemia to those found in the copper-deficiency anemia.

=> d his

(FILE 'HOME' ENTERED AT 14:26:59 ON 16 NOV 1999)

FILE 'MEDLINE, CANCERLIT, BIOSIS, EMBASE, SCISEARCH' ENTERED AT 14:28:40 ON 16 NOV 1999
L1 2 S INPROL
L2 0 S FPHFDLSHGSAQVS OR PHE-PRO-HIS-PHE-ASP-LEU-SER-HIS-GLY-SER-ALA
L3 3111 S HEMOGLOBIN AND (STEM CELL OR HEMATOPOIE? OR PROGENITOR(W)CELL
L4 1002 S L3 AND (STIMULAT? OR PROLIFER?)
L5 466 DUP REM L4 (536 DUPLICATES REMOVED)
L6 790 S L3 AND (INHIB? OR REDUC? OR ABROGAT? OR ANTAGON?)
L7 381 DUP REM L6 (409 DUPLICATES REMOVED)
L8 80 S HEMOGLOBIN AND ((STEM CELL OR HEMATOPOIE? OR PROGENITOR(W)CEL
L9 31 DUP REM L8 (49 DUPLICATES REMOVED)

=> logoff

ALL L# QUERIES AND ANSWER SETS ARE DELETED AT LOGOFF
LOGOFF? (Y)/N/HOLD:y

COST IN U.S. DOLLARS	SINCE FILE ENTRY	SESSION	TOTAL
FULL ESTIMATED COST		50.68	51.13

STN INTERNATIONAL LOGOFF AT 15:01:19 ON 16 NOV 1999